Biopsy specimen appearances of ischaemic gastritis in splanchnic arterial insufficiency

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Abstract

A 74 year old man presented with a one month history of epigastric discomfort, anorexia, weight loss, and postprandial vomiting. The diagnosis of ischaemia was made on endoscopic biopsies from the stomach and duodenum. He was too ill for major vascular surgery and died eight days after admission. Postmortem examination confirmed the diagnosis of splanchnic arterial insufficiency caused by atheroma and thrombosis. Ischaemic gastritis is rare but could easily be missed in unrepresentative biopsy specimens. Prompt diagnosis with revascularisation surgery is the only hope for long term survival.

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Ischaemic gastritis caused by thromboatheromatous disease of the aorta and main splanchnic arteries is extremely uncommon because of the rich collateral blood supply of the stomach. The few cases recorded (all heavy smokers) generally had similar endoscopic appearances[1–6] with multiple aphthoid ulcers superimposed on a friable congested mucosa. In two cases[4 5] the endoscopic appearances returned to normal following revascularisation. Biopsies, when taken, have usually not contributed to the diagnosis,[1 2 5] although some have been suggestive of ischaemia, but not illustrated.[6] The gross postmortem appearances of the stomach[7] are similar to the endoscopic appearances with multiple ulcers of varying size in a haemorrhagic mucosa. Atheromatous emboli in arteries in the stomach wall have been described as causing ischaemic gastric necrosis in postmortem histology[7] and in a surgical resection specimen.[8]

In the case described here the endoscopic biopsy appearances were sufficiently distinctive to make a diagnosis of ischaemia, the cause of which became apparent over the subsequent few days and at postmortem.

Ischaemic ulceration of the stomach can also be caused by polyarteritis nodosa and leucocytoclastic vasculitis,[9] and recently some ischaemic changes have been described due to secondary arterial changes in portal hypertension.[10]

Case report

A 74 year old man was admitted because of frequent vomiting associated with periumbilical pain and some diarrhoea. For the previous month he had epigastric discomfort, no appetite, profound weight loss, and intermittent vomiting after meals. In the past a right inguinal hernia had been repaired twice; he was a heavy smoker, up to 60 cigarettes daily throughout his adult life.

On examination he was pale, dehydrated, and cachectic with abdominal tenderness and guarding. A bruit was heard over the renal arteries. Laboratory investigations were normal apart from a total white cell count of 20×10^9/l with a polymorph leucocytosis, and slightly raised serum amylase of 282 U/l (normal < 220). Plain abdominal radiography and abdominal ultrasound showed some fluid filled, non-dilated small bowel loops, but nothing else of relevance. Intra-abdominal sepsis was suspected and he was treated with antibiotics, intravenous fluids, and nasogastric suction.

Endoscopy of the oesophagus the following day appeared normal; 300 ml of faeculent fluid was aspirated from the stomach revealing severe gastritis and duodenitis, and biopsy specimens were taken.

Two days later the patient developed a cold numb left foot with absent left femoral pulse. He had a left femoral embolectomy under epidural anaesthesia, but because of poor proximal pulses he required a femorofemoral crossover graft. This was successful, but then the endoscopic biopsy reports were received showing ischaemic gastritis and duodenitis. A presumed diagnosis of mesenteric ischaemia was made but he was too ill for major surgery. He developed increasing abdominal tenderness with absent bowel sounds and died eight days after admission.

Pathology

Two biopsy specimens from the greater curve of the body of the stomach, and two from the second part of the duodenum were investigated. Both gastric specimens showed focal full thickness coagulative necrosis of the mucosa with sloughing of the necrotic debris. Although the necrotic areas contained some neutrophil polymorphs, it was possible to identify the ghost outline of glands and pits represented by vertical columns of necrotic epithelial cells containing pyknotic nuclei (fig 1). The adjacent mucosa showed some atrophy of the cells lining the glands, with hyalinisation of the intervening stroma, consistent with ischaemia. Some of the mucosa was normal with no significant inflammation, and the muscularis mucosae was normal.

The duodenal biopsy specimens also showed focal full thickness mucosal necrosis with atrophy of adjacent villi, together with a pseudomembranous exudate of fibrin and polymorphs (fig 2). Elsewhere the mucosa was normal, as was the muscularis mucosae and submucosal blood vessels.

The combination of coagulative-type necrosis, ischaemic atrophy of the adjacent mucosa,

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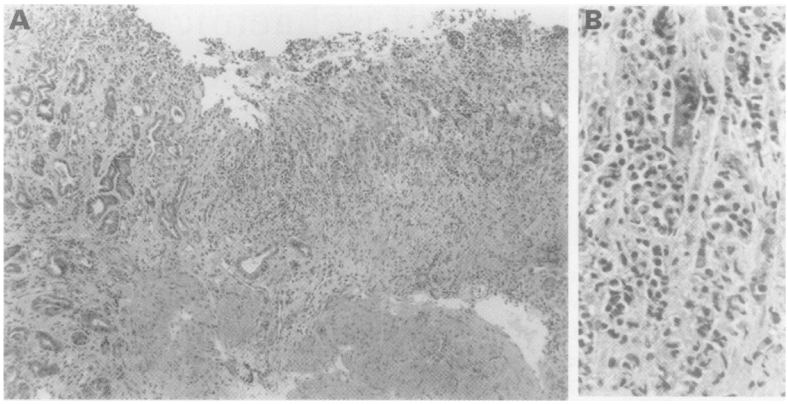

Figure 1 Gastric biopsy (haematoxylin and eosin stain); (A) low power; (B) high power. There is full thickness mucosal necrosis with vertical columns of necrotic epithelial cells admixed with some neutrophil polymorphs (B). The capillaries are congested. The adjacent mucosa shows ischaemic atrophy.

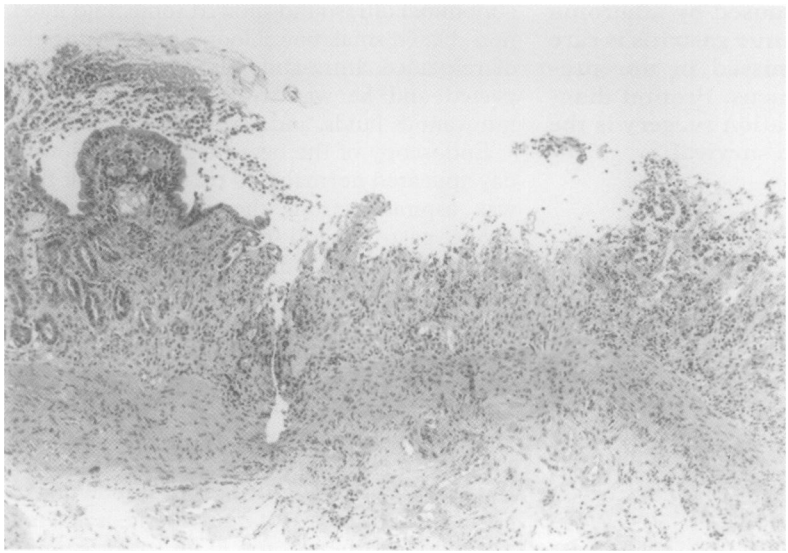

Figure 2 Duodenal biopsy (haematoxylin and eosin stain). There is full thickness mucosal necrosis with a surviving atrophic villus. There is a pseudomembrane of fibrin and polymorphs on the left.

the multiplicity of ulcers, and the chronic history were regarded as diagnostic of ischaemic ulceration.

At postmortem examination there was purulent peritonitis over some loops of small bowel showing haemorrhagic infarction. The distal half of the stomach, the remainder of the small bowel, and the ascending colon were dusky purple and had several ulcers. There were also a few ischaemic ulcers in the transverse colon. The oesophagus, proximal stomach, distal colon, and rectum were normal. The abdominal aorta was severely atheromatous and there was a disc of mural thrombus 3 cm diameter covering the origins of the coeliac and superior mesenteric arteries; this extended into and occluded the proximal 0.5 cm of both arteries, which were narrowed by atheroma. The origin of the inferior mesenteric artery was severely narrowed by atheroma, and the left common iliac artery was occluded by thrombus and atheroma. The heart (400 g) had moderate coronary artery atheroma; the myocardium was normal and there were no endocardial thrombi or vegetations. The liver, gall bladder, and pancreas were normal, and the kidneys (280 g) and renal arteries were unremarkable.

Discussion

Most erosions and ulcers of the stomach, regardless of cause, have a final common pathway of acid and pepsin digestion that results in progressive liquefaction necrosis of the surface. However, the gastric erosion illustrated here shows full thickness coagulative necrosis of the mucosa with sloughing of the necrotic debris, there is also some ischaemic atrophy of the adjacent mucosa. One gastric biopsy included some normal mucosa, and the focal distribution of the ischaemic changes may explain the non-contributory findings in two of the previous cases in which biopsy was done.[1 2]

Ischaemic gastritis caused by splanchnic arterial insufficiency is extremely rare because of the rich collateral blood supply of the stomach, and is only seen when at least two of the three main splanchnic arteries are occluded or severely stenosed.[5 6] The main blood supply of the stomach is from branches of the coeliac artery, which anastomose freely with each other, and there is collateral supply from the first branch of the superior mesenteric artery. However, there is also systemic collateral circulation from the oesophageal arteries, and should it arise directly from the aorta, from the left inferior phrenic artery.

This systemic collateral arterial supply probably explains the focal nature of the ischaemic necrosis in this case, and the sparing of the proximal stomach.

Ischaemic ulceration of the stomach has also been reported caused by polyarteritis nodosa and leucocytoclastic vasculitis,[9] and rarely in portal hypertension,[10] but none of these factors were relevant in our case.

Splanchnic arterial insufficiency causing ischaemic gastritis and duodenitis is a rare disease confined to heavy cigarette smokers, which has a grave prognosis.[6] Revascularisation is occasionally successful[4-6] provided that the patients are not too ill for major vascular surgery.

The various features enabling a biopsy diagnosis of ischaemic gastritis and duodenitis to be made in this case were the chronic history, coagulative-type necrosis in the ulcers, the presence of similar ulcers in the stomach and duodenum, and the ischaemic atrophy of the adjacent mucosa.

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